

FILE 'MEDLINE, BIOSIS, CAPLUS' ENTERED AT 15:52:56 ON 10 OCT 2003

L2 250 S CIS-HYDROXYPROLINE
L3 250 S L2 AND HYDROXYPROLINE
L4 0 S L3 AND ALLO-L-HYDROXYPROLINE
L5 0 S L3 AND ALLO-HYDROXY-L-LPROLINE
L6 0 S L3 AND ALLO-HYDROXY-L-PROLINE
L7 12 S 4-CIS-HYDROXY-L-PROLINE
L8 7 S L2 AND ASSAY

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L Number	Hits	Search Text	DB	Time stamp
1	53	cis-hydroxyproline	USPAT; US-PGPUB; EPO; JPO; DERWENT	2003/10/14 12:49

L1 ANSWER 1 OF 1 REGISTRY COPYRIGHT 2003 ACS on STN
RN 618-27-9 REGISTRY
CN L-Proline, 4-hydroxy-, (4S)- (9CI) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN L-Proline, 4-hydroxy-, cis-
CN Proline, 4-allo-hydroxy- (7CI)
CN Proline, 4-allo-hydroxy-, L- (8CI)
OTHER NAMES:
CN (2S,4S)-4-Hydroxyproline
CN (S)-allo-Hydroxyproline
CN 4 (S)-Hydroxy-2 (S)-pyrrolidinecarboxylic acid
CN 4-cis-Hydroxy-L-proline
CN allo-4-Hydroxyproline
CN allo-Hydroxy-L-proline
CN allo-L-Hydroxyproline
CN cis-4-Hydroxy-L-proline
CN cis-4-Hydroxyproline
CN cis-Hydroxyproline
CN L-allo-4-Hydroxyproline
CN L-allo-Hydroxyproline
CN L-Allohydroxyproline
CN L-cis-4-Hydroxyproline
CN L-Proline, allo-hydroxy-
CN NSC 206274
FS STEREOSEARCH
DR 3398-20-7, 30724-02-8
MF C5 H9 N O3
CI COM
LC STN Files: AGRICOLA, ANABSTR, BEILSTEIN*, BIOBUSINESS, BIOSIS, CA,
CAOLD, CAPLUS, CASREACT, CHEMCATS, CHEMINFORMRX, CHEMLIST, CSCHEM,
DETERM*, GMELIN*, HODOC*, MRCK*, NAPRALERT, NIOSHTIC, TOXCENTER,
USPAT2, USPATFULL
(*File contains numerically searchable property data)
Other Sources: EINECS**, NDSL**, TSCA**

L Number	Hits	Search Text	DB	Time stamp
1	0	("hydroxyproline").PN.	USPAT; US-PGPUB; EPO; JPO; DERWENT	2003/10/10 15:36
2	0	("cis-Hyp").PN.	USPAT; US-PGPUB; EPO; JPO; DERWENT	2003/10/10 15:36
3	7580	hydroxyproline	USPAT; US-PGPUB; EPO; JPO; DERWENT	2003/10/10 15:37
4	53	cis-hydroxyproline	USPAT; US-PGPUB; EPO; JPO; DERWENT	2003/10/10 15:37
5	27	cis-hydroxyproline and detect\$3 o' neal@X	USPAT; US-PGPUB; EPO; JPO; DERWENT	2003/10/10 15:38

(FILE 'HOME' ENTERED AT 15:50:54 ON 10 OCT 2003)

FILE 'MEDLINE, BIOSIS, CAPLUS' ENTERED AT 15:51:00 ON 10 OCT 2003

FILE 'REGISTRY' ENTERED AT 15:51:07 ON 10 OCT 2003

L1 1 S CIS-HYDROXYPROLINE

FILE 'MEDLINE, BIOSIS, CAPLUS' ENTERED AT 15:52:56 ON 10 OCT 2003

L2 250 S CIS-HYDROXYPROLINE

L3 250 S L2 AND HYDROXYPROLINE

L4 0 S L3 AND ALLO-L-HYDROXYPROLINE

L5 0 S L3 AND ALLO-HYDROXY-L-LPROLINE

L6 0 S L3 AND ALLO-HYDROXY-L-PROLINE

L7 12 S 4-CIS-HYDROXY-L-PROLINE

ANSWER 2 OF 7 MEDLINE on STN
AN 89225504 MEDLINE
DN 89225504 PubMed ID: 2469316
TI Matrix control of tumor angiogenesis.
AU Reilly W; McAuslan B R
CS CSIRO Division of Molecular Biology, North Ryde, NSW, Australia.
SO ADVANCES IN EXPERIMENTAL MEDICINE AND BIOLOGY, (1988) 242 221-7.
Journal code: 0121103. ISSN: 0065-2598.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 198905
ED Entered STN: 19900306
Last Updated on STN: 20000303
Entered Medline: 19890530
AB Endothelial cell migration is a key feature of angiogenesis. Epidermal Growth Factor (EGF) or Tumor Angiogenesis Factor (TAF) induce cell migration and angiogenesis. When the matrix components, collagen or fibronectin, were used as a substratum in the phagokinetic assays, EGF- or TAF-induced cell migration was inhibited. It has been proposed that TAF activates cellular protease causing the matrix degradation that is evident during neovascularization in vitro. If such degradation leads to cell migration and angiogenesis, then other agents that interfere with the synthesis or assembly of matrix components should stimulate cell migration and angiogenesis. The proline analogues *cis* hydroxyproline, azetidine and dehydroproline are known modulators of cellular collagen synthesis. At optimal concentration ($10(-5)$ M) these analogues caused 3-fold increases in endothelial cell migration rates in vivo as tested by a subcutaneous implant assay. We conclude from these studies that: (i) matrix components control cellular migration rates; high concentration of collagen or fibronectin inhibit angiogenically active inducers of endothelial cell migration. (ii) Intracellular modulation of synthesis of collagens leads to angiogenesis by stimulating cell migration. These findings relate to tumor angiogenesis and that TAF might trigger angiogenesis either by activation of latent proteases or by some modification of matrix assembly during synthesis that affects cell adhesion and migration.

L8 ANSWER 1 OF 7 MEDLINE on STN
AN 97192893 MEDLINE
DN 97192893 PubMed ID: 9040485
TI **cis-Hydroxyproline** inhibits proliferation, collagen synthesis, attachment, and migration of cultured bovine retinal pigment epithelial cells.
AU Yoo J S; Sakamoto T; Spee C; Kimura H; Harris M S; Hinton D R; Kay E P;
Ryan S J
CS Doheny Eye Institute, University of Southern California School of Medicine, Los Angeles 90033, USA.
NC EY01545 (NEI)
EY03040 (NEI)
SO INVESTIGATIVE OPHTHALMOLOGY AND VISUAL SCIENCE, (1997 Feb) 38 (2) 520-8.
Journal code: 7703701. ISSN: 0146-0404.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199703
ED Entered STN: 19970321
Last Updated on STN: 19970321
Entered Medline: 19970311
AB PURPOSE: Proliferative vitreoretinopathy (PVR) is characterized by the proliferation and migration of retinal pigment epithelial (RPE) and other cells into the vitreous cavity. The PVR membrane formation also is associated with collagen production by RPE. The authors examined the effect of a proline analog, **cis-hydroxyproline** (CHP), on proliferation, collagen synthesis, attachment, and migration of bovine RPE in vitro. METHODS: The effect of CHP on cell proliferation was determined as a function of dosage and days in culture by counting the cell numbers on days 3, 6, and 9. Collagen synthesis was determined by trichloroacetic acid precipitation of the radiolabeled samples before and after bacterial collagenase digestion. The attachment assay involved type I collagen or fibronectin substrates or both (2.5 micrograms/well). For migration experiments, RPE cells were removed from a defined area of a confluent culture, and migration was quantitated by counting the number of cells migrating into the denuded area over 30 hours. RESULTS: The addition of CHP inhibited RPE proliferation in both a dose- and a time-dependent manner; collagen synthesis, attachment, and migration also were inhibited by CHP in a dose-dependent manner. When the culture plates were coated with collagen, < 100 micrograms/ml of CHP had no effect on cell attachment. Higher doses of CHP resulted in mild inhibition of attachment on collagen-coated plates. Simultaneous addition of L-proline to the cultures resulted in blockade of these inhibitory effects on proliferation, collagen synthesis, attachment, and migration. CONCLUSIONS: The results show that RPE functions critical to the development of PVR are inhibited by CHP, suggesting the possibility that this drug may